predicted DH domains are expressed during the first 24 h of embryogenesis. For 23 of these RhoGEFs, we designed splice-blocking morpholinos (MOs) to block inclusion of one exon and introduce a frame shift upstream of the DH domain, leading to nonsense-mediated decay in three out of three instances tested. To maintain a high-throughput approach and to focus our analysis on morphogenesis, we developed a time-lapse system that traces the parallel growth of over 50 embryos. In this way, we have identified three RhoGEFs that are essential for epiboly movements and yolk cell integrity, and a fourth RhoGEF that is critical for survival beyond the bud stage. Defects in F-actin distribution for these four morphants are consistent with their phenotypes. Using 5′ and 3′ RACE, we have obtained full-length clones for these four genes and biochemical assays to identify their RhoGTPase targets are being pursued.

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Msx1 and *Msx2* are essential for myocardial patterning and morphogenesis of the outflow tract and atrioventricular cushions

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Combined deficiencies of the homeobox genes *Msx1* and *Msx2* in mice have been shown to cause a broad spectrum of cardiac anomalies overlapping with those seen in human patients of Wolf–Hirschhorn syndrome. Here we investigate the cellular mechanisms that contribute to these cardiac defects. In the *Msx1–Msx2* double mutant anterior heart field progenitor cells, there was normal expression of early markers *Isl1* and *Mef2c*, yet reduced expression of later markers *Hand1*, *Hand2*, *Fgf10* and *Pitx2*. Normal dextral cardiac looping in the *Msx1–Msx2* double mutants further supports that *Msx1* and *Msx2* function primarily in post-looping morphogenesis. Misexpressed *Pitx2* was also observed in the double mutant atrial myocardium and is associated with mispatterning of the atrial myocardium as well as atrial septal defect. In addition, we found excessive proliferation of cardiac neural crest, endothelial and myocardial cells in the double mutant outflow tract between E10 and E11, which is associated with increased Bmp signaling and reduced $p27^{KIP1}$ expression and may further contribute to the outflow malalignment defects. Moreover, in the double mutant atrioventricular cushions, reduced levels of *Bmp2, Bmp4* and *Pitx2* caused impaired endothelial-to-mesenchymal transdifferentiation and hypoplastic atrioventricular valves. In summary, our findings suggest that *Msx1* and *Msx2* are key regulators of post-looping development of the outflow tract myocardium and endocardium, atrial myocardium as well as atrioventricular cushions and valves.

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CASTOR is fundamentally required for early embryogenesis in *Xenopus*

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We have identified a homologue of *Drosophila* Castor, a zinc finger transcription factor, in both *Xenopus laevis* and *Xenopus tropicalis*. *Drosophila* Castor has been shown to be one of the last transcription factors expressed by late-born neuroblasts prior to their differentiation, thereby specifying neuronal cell fate. In addition, Castor has been identified in mouse, where it is also highly expressed in the developing heart. We have gone on to characterize *Xenopus* Castor expression by in situ hybridization revealing Castor to be expressed in the developing heart and CNS. RLM-RACE was used to determine 5′ cDNA sequence of Castor. Results from these studies have shown the presence of two Castor transcripts, both with unique transcriptional and translational initiation sites. To determine the precise requirement for Castor, we designed morpholinos in order to deplete each alternate transcript individually and in combination. Results from these studies show a fundamental embryonic requirement for CASTOR in both the heart and in the nervous system. In the cardiac lineage, as shown by histology, TEM, in situ, and antibody staining, CASTOR is required during stages coinciding with early cardiac differentiation. Since depletion of either Castor transcript leads to cardiac abnormalities, our results suggest that both CASTOR proteins are required for early steps of cardiogenesis.

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Development of adult abdominal muscle innervation in *Drosophila*

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In holometabolous insects such as *Drosophila*, the transition from larval to adult body form is characterized by radical transformations. Behaviorally, a crawling animal is transformed into one that is capable of flight. Thus, two distinct motor systems are generated during the life-cycle of the animal. Formation of the adult motor system involves remodeling of persistent larval motor neurons, which innervate muscle fibers that are mostly generated anew. We are using the adult abdominal muscles as a model to study remodeling and developmental plasticity during metamorphosis. Preliminary work in our laboratory has characterized the neuromuscular junctions of abdominal muscles (Hebbar, et al., J. Neurobio., in press). In order to correlate the remodeling of motor neurons in the central nervous system with the changes in innervation and motor units at the periphery, motor neuron-specific Gal4 drivers are being used to identify the location of cell bodies in the CNS as well as their characteristic projections in the periphery. This will be